Anatomy and Biomechanics of the Lateral Side of the Knee

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Abstract: The posterolateral corner (PLC) of the knee is a critical element for a functional lower extremity. It consists of an array of complex ligamentous and musculotendinous structures. The primary function of the PLC is to resist varus and external rotation and posterior translation of the tibia. Injuries to these structures can cause significant disability and compromise activities of daily living and work, recreational, and sporting activities. A thorough understanding of the complex anatomy and biomechanics of the PLC will aid the clinician in this challenging diagnostic and therapeutic problem. The first section of this paper describes the anatomy of the PLC of the knee focusing on the intricate insertion sites of the individual structures. The second section discusses how the anatomy influences the biomechanics of the PLC.

Key Words: knee, anatomy, biomechanics, ligament, posterolateral corner, popliteus

The posterolateral corner (PLC) of the knee is a critical element for a functional lower extremity. It consists of an array of complex ligamentous and musculotendinous structures. The primary function of the PLC is to resist varus and external rotation and posterior translation of the tibia. Injuries to these structures can cause significant disability and compromise activities of daily living and work, recreational, and sporting activities. A thorough understanding of the complex anatomy and biomechanics of the PLC will aid the clinician in this challenging diagnostic and therapeutic problem. The first section of this paper describes the anatomy of the PLC of the knee focusing on the intricate insertion sites of the individual structures. The second section discusses how the anatomy influences the biomechanics of the PLC.

ILIOTIBIAL BAND

The iliotibial band is a thick fascial sheath extending over the tensor fasciae latae muscle along the lateral aspect of the thigh. This structure originates from the anterior superior iliac spine and the anterior part of the external lip of the iliac crest. It inserts onto the anterolateral aspect of the lateral tibial plateau. Its insertion on the tibia was originally described by Gerdy and later popularized by Segond as the “tubercle of Gerdy.” Today it is referred to as “Gerdy tubercle.”1

The iliotibial band is divided into superficial, deep, and capsulo-osseous layers. The superficial layer is first encountered after dissecting through the subcutaneous tissues on the lateral aspect of the leg. After splitting the first fascial layer (superficial layer) of the iliotibial band, deeper fibers intimately adhere to the lateral supracondylar tubercle of the femur and blend into the lateral intramuscular septum. These layers, now called the deep and capsulo-osseous layers of the iliotibial band, are commonly known as “Kaplan fibers” (Fig. 1). An anterior portion of the iliotibial band, known as the iliopatellar band, curves anteriorly to insert onto the lateral aspect of the patella.1,2

The deep layer of the iliotibial band is visualized beginning 6 cm proximal to the lateral femoral epicondyle, at the termination of the lateral intramuscular septum. It covers a triangular-shaped area over the lateral supracondylar face of the distal femur. It connects the medial border of the superficial iliotibial layer to the distal termination of the lateral intramuscular septum of the distal femur.3

Medial and distal to the deep layer, the capsulo-osseous layer originates from the region of the lateral intramuscular septum and the fascia over the posterolateral aspects of the lateral gastrocnemius and plantaris

FIGURE 1. Retraction of the superficial layer of the iliotibial tract reveals the deep (DITT) and capsulo-osseous (COITT) layers. G, Gerdy tubercle. Reprinted with permission.16
muscles. Along the lateral aspect of the knee, this structure blends with the short head of the biceps femoris in a region known as the confluence of the short head of the biceps femoris and the capsulo-osseous layer. The capsulo-osseous layer functions as an anterolateral ligament of the knee, as it forms a sling over the lateral femoral condyle.\(^2,5\) Distally, it inserts onto the lateral distal femoral condyle.\(^2,5\) This layer, reinforced by the deep layer, tethers the superficial layer to the distal-lateral aspect of the femur, forming a sling behind the lateral femoral condyle. Historically, this lateral sling of tissue is what most surgeons attempted to reconstruct in extra-articular anterior cruciate ligament (ACL) reconstructions.

**BICEPS FEMORIS**

The biceps femoris is a fusiform muscle that contains 2 heads, long and short. The long head of the biceps femoris muscle originates from the ischial tuberosity and is part of the common hamstring origin. It is innervated by the tibial division of the sciatic nerve. The short head of the biceps femoris muscle originates just medial to the linea aspera of the distal femur, and is innervated by the peroneal division of the sciatic nerve. Both heads flex the leg at the knee joint and laterally rotate the leg when it is flexed.\(^7\)

The long head of the biceps has 5 major insertions at the knee. It divides into 2 tendinous components called the direct and anterior arms, and 3 fascial components called the reflected arm and, anterior and lateral, aponeurotic expansions. The most important of these structures are the direct and anterior arms, and the lateral aponeurotic expansion. The direct arm inserts onto the lateral aspect of the fibular styloid. The anterior arm courses lateral to the fibular collateral ligament (FCL) and inserts onto the lateral tibial plateau.\(^6\) A small biceps femoris bursa separates this arm from the FCL.\(^8\) The lateral aponeurotic expansion connects the long and short heads of the biceps femoris to the posterolateral aspect of the FCL.\(^5,6\)

The short head of the biceps femoris muscle divides into 6 components. These components consist of direct, capsular, and anterior tendinous arms, along with 3 nontendinous insertions. The 3 nontendinous attachments include a muscular insertion onto the long head of the biceps femoris tendon, a lateral aponeurotic expansion attaching both the short and long heads to the posteromedial aspect of the FCL, and a third insertion, just lateral to the capsular arm, forming a confluence with the capsulo-osseous layer of the iliotibial tract. This confluence with the capsulo-osseous layer forms an intimate attachment between the fascia of the iliotibial band and the biceps femoris.\(^5,6\) (Fig. 2).

The most important insertions of the short head of the biceps femoris are the 3 tendinous arms. They are most easily identified at the level of the fibular head. The direct arm is the least important of the 3 and attaches to the posterolateral aspect of the fibular styloid.

**FCL**

The FCL, also known as the lateral collateral ligament, is the primary static stabilizer to varus opening of the knee in the initial 0-degree to 30-degree arc of knee flexion. With knee flexion beyond 30 degrees, the FCL becomes slightly lax.\(^4\) In addition, this ligament provides resistance to external rotation of the tibia, primarily near extension.

The femoral origin of the FCL is extracapsular and easily identified by palpating the lateral epicondyle. LaPrade et al.\(^9\) have shown that the femoral insertion of the FCL consistently lies 1.4 mm proximal and 3.1 mm
posterior to the lateral epicondyle. This location may be palpated as a small bony depression formed between the lateral epicondyle and the suprapatellar process.

The FCL is approximately 70 mm long and its primary distal insertion site is the lateral aspect of the fibular head. During a surgical approach, the FCL can be easily identified through a small 3-cm incision made parallel to the fibers of the long head of the biceps femoris tendon. This incision is made superficial to the proximal extent of the fibular head and should release the insertion of the anterior arm of the long head of the biceps femoris tendon. This incision should readily expose the biceps bursa, which forms an inverted “J” shape around the anterior and anteromedial portions of the FCL. The biceps bursa encapsulates the distal 25% of the FCL. The long head of the biceps femoris tendon forms the lateral border of the biceps bursa.

The fibular insertion of the FCL lies 8.2 mm posterior to the anterior border of the fibula, and 28.4 mm antero-inferior to the proximal tip of the fibular styloid. On average, this comprises 38% (13.9 mm) of the total width of the fibular head, which measures 36.6 mm anterior to posterior.

POPLITEUS MUSCLE/POPLITEUS COMPLEX

The popliteus is an obliquely oriented muscle that originates from the postero-medial aspect of the proximal tibia. It forms the floor of the inferior part of the popliteal fossa and is innervated by the tibial nerve. The muscle gives rise to the popliteus tendon at the lateral one-third of the popliteal fossa in the PLC. The average length of the popliteus tendon is 54.5 mm. The tendon continues proximally, through the popliteal hiatus in the coronary ligament, at which point it becomes intra-articular, and inserts onto the lateral femoral condyle. This popliteal hiatus is also known as the “bare area of the lateral meniscus.”

The popliteus is a dynamic internal rotator of the tibia and is believed to contribute to the dynamic stability of the lateral meniscus. To facilitate these functions, it has multiple insertions on the postero-lateral aspect of the knee. The popliteus muscle-tendon unit and its multiple ligamentous connections from the tendon to the fibula, tibia, and meniscus are known as the popliteus complex. Each of these ligamentous connections have been further clarified. The ligamentous insertion on the fibula is comprised of the anterior and posterior popliteofibular ligaments. Historically, these 2 ligaments were collectively known as the arcuate ligament. The popliteal insertion onto the tibia is known as the muscular aponeurotic attachment. The popliteal connections to the lateral meniscus are known as the 3 popliteomeniscal fascicles. This complex provides dynamic and static stability to the knee primarily in response to external tibial rotation.

The femoral insertion of the popliteus tendon is intra-articular and requires an arthrotomy for clear identification. During a surgical approach, the arthrotomy should begin just distal to the lateral epicondyle, run parallel to the FCL, and end at around the level of the lateral meniscus. The popliteus should be easily identified in the popliteal sulcus of the femur. The insertion of the popliteus is consistently found at the most anterior 1/5 and proximal 1/2 of the sulcus. The average cross-sectional area of the popliteus sulcus is 3.4 cm²; 0.59 cm² of the popliteal sulcus is used for the footprint of the popliteus tendon.

Numerous researchers have found that the popliteus tendon insertion site was consistently anterior to the FCL along the lateral aspect of the lateral femoral condyle (Fig. 4). Specifically, LaPrade et al. located the popliteus insertion site 18.5 mm distal and anterior to the femoral insertion of the FCL, and 15.8 mm distal and anterior to the lateral epicondyle of the femur (Fig. 5). Furthermore, they noted that the popliteus tendon did not completely enter the confines of the popliteal sulcus until the knee was flexed to an average of 112 degrees. In knee flexion less than 112 degrees, the tendon subluxed anteriorly out of the sulcus (Fig. 6).

As the popliteus tendon courses proximal and laterally, it gives off 3 branches which contribute to the dynamic stability of the lateral meniscus. These are known as the popliteomeniscal fascicles.

The first meniscal fascicle is the popliteus muscle’s aponeurotic attachment. This meniscal aponeurosis is an attachment of the popliteus muscle to the posterior capsule and lateral meniscus. This structure, initially described by Last, was termed the postero-inferior popliteomeniscal fascicle by Staubbli and Birrer. Located medial to the popliteus tendon, this popliteomeniscal fascicle provides a posterior tibial attachment for the lateral meniscus.

The postero-superior popliteomeniscal fascicle arises from the medial superior surface of the popliteus tendon as it penetrates the posterior capsule. It blends into the
The posterior horn of the lateral meniscus and the meniscofemoral portion of the posterior capsule of the joint. The posteroinferior popliteomeniscal fascicle forms from the popliteus muscle aponeurosis, just medial to the tendon, whereas the postero-superior popliteomeniscal fascicle forms from the popliteus tendon. The ligament of Wrisberg bisects these 2 posterior popliteomeniscal fascicles as it connects the posterior horn of the lateral meniscus to the medial femoral condyle, providing additional stability to the lateral meniscus.

The last fascicle, the antero-inferior popliteomeniscal fascicle, originates the furthest distally along the popliteus tendon. From the anterior edge of the popliteus tendon, it blends into the middle third of the lateral meniscus forming the anterior boundary of the popliteal hiatus.

The biomechanical significance of each of the 3 individual popliteomeniscal fascicles is not completely understood. However, it is known that together they stabilize the lateral meniscus and prevent medial entrapment of the meniscus during varus forces on the knees. These functions are complicated by the need to permit the large excursion of the lateral meniscus over the chondral surfaces.

The popliteofibular ligament is the second most prominent structure in the PLC, with the most prominent being the popliteus tendon. The anatomy and function of the popliteofibular ligament is becoming more defined.

The popliteofibular ligament is a static stabilizer of the lateral and posterolateral knee, resisting varus, external rotation, and posterior tibial translation. A force of 298 N is required to cause failure of this structure. Alternatively, rather than an intrasubstance tear, the posterior division may avulse a fragment of the fibular head in what is known as an arcuate fracture. Arcuate fractures are often associated with cruciate ligament injuries. Due to their roles as both static and dynamic restraints, the popliteus and popliteofibular ligaments are vital components of any posterolateral reconstructive procedure.

Directly posterior to the popliteofibular ligament courses the inferior lateral geniculate artery. This artery...
originates off the popliteal artery and courses along the posterior joint capsule just proximal to the superior aspect of the lateral meniscus. As it courses laterally, it bisects the popliteofibular ligament and the fabellofibular ligament (capsular arm of the short head of the biceps femoris) as they attach to the fibular styloid. The fabellofibular ligament lies anterior and the fabellofibular ligament lies posterior to the inferior lateral geniculate artery where it crosses the posterior aspect of the fibular styloid.  

The final component of the popliteus complex is its popliteotibial insertion. This is a direct attachment extending from the anterior surface of the popliteus muscle to an area just lateral to the fovea of the posterior cruciate ligament (PCL). It inserts on the inferior surface of the lateral meniscus and on the posterior capsule. The insertion on the posterior capsule lies medial to the popliteal tendon, an area devoid of a coronary ligament attachment.  

**LATERAL GASTROCNEMIUS TENDON**

The lateral gastrocnemius tendon originates at or near the supracondylar process of the distal femur. In this region, this tendon can be easily identified by blunt dissection medial and distal to the long head of the biceps femoris in the interval between the lateral gastrocnemius and the soleus muscle. On average, it attaches 13.8 mm posterior to the FCL, 28.4 mm posterior to the popliteus tendon insertion, and is nearly inseparable from the meniscofemoral portion of the posterior capsule. At the level of the fibular styloid, the lateral head of the gastrocnemius blends with the popliteofibular ligament, providing varying degrees of additional postero-lateral stability. 

**JOINT CAPSULE/MIDTHIRD LATERAL CAPSULAR LIGAMENT**

The joint capsule can be divided into superficial and deep laminae. The 2 laminae become confluent anterior to the overlying iliotibial band. The inferior lateral geniculate artery, traveling through an anterior truncated space between the deep and superficial laminae, always separates these 2 capsular structures.  

The superficial lamina, which is the original capsule embryologically, encompasses the FCL and ends posteriorly at the fabellofibular ligament.  

The deep capsular lamina is phylogenetically younger and is a result of the fibula receding from the lateral femur. It extends posterolaterally and forms the coronary ligament and the hiatus for the popliteus tendon. The deep lamina travels along the lateral meniscus
and spans from the junction of the popliteus muscle and tendon to its termination at the popliteofibular ligament. In the cranial to caudal direction, it extends from the femur to the fibula. An in vivo anatomic and magnetic resonance imaging study demonstrated that the most inferior extent of the capsular reflection of the knee is along the posterior fibula. The most extensive capsular reflection was less than 14 mm inferior to the subchondral bone of the proximal tibia. This anatomic finding has lent credence to the recommendation of placing fine wires for circular fixators no closer than 15 mm inferior to the articular surface to minimize the incidence of iatrogenic knee sepsis secondary to pin tract infection.

The joint capsule of the knee can be divided into 3 sections in the anterior to posterior direction: anterior, lateral, and posterior. The anterior section extends from the patella tendon to the anterior border of the popliteus tendon’s insertion on the femur. It is adherent to the patellar fat pad, intermeniscal ligament, and anterior horn of the lateral meniscus. The lateral capsule extends from the anterior border of the popliteus tendon’s insertion on the femur to the lateral gastrocnemius attachment. The posterior capsule is attached to the femur, proximal to the articular margin of the lateral femoral condyle.

From medial to lateral, the posterior capsule is covered by the muscular origins of the plantaris and lateral gastrocnemius muscle and tendon. Distally, it blends with the musculotendinous junction of the popliteus and the posterior division of the popliteofibular ligament.

The midthird lateral capsular ligament is a thickening of the lateral capsule of the knee. It is divided into 2 components: the meniscofemoral and meniscotibial components. It is thought to be homologous to the deep medial collateral ligament on the medial aspect of the knee. The meniscofemoral component extends from the femur down to the meniscus and the meniscotibial component extends up from the tibia to the meniscus. Anterior to the popliteal hiatus, the lateral meniscus is stabilized by the meniscotibial portion of the midthird lateral ligament. Biomechanically, this structure is thought to be an important secondary stabilizer to varus instability.

**CORONARY LIGAMENT OF THE LATERAL MENISCUS**

The coronary ligament of the lateral meniscus lies posterior to the midthird lateral capsular ligament. It is the meniscotibial portion of the posterior joint capsule extending from the anterior margin of the popliteal hiatus to the lateral aspect of the posteriorinferior popliteome-niscal fascicle. It secures the posterior horn of the lateral meniscus to the tibia. The coronary ligament is important clinically in providing resistance to hyperextension and posterolateral rotation of the tibia.

**OBLIQUE POPLITEAL LIGAMENT**

The oblique popliteal ligament (or ligament of Winslow) is formed by the coalescence of the oblique popliteal expansion of the semimembranosus and the capsular arm of the posterior oblique ligament. These 2 structures originate from the medial side of the knee, merge anterior to the medial head of the gastrocnemius, and form the oblique popliteal ligament. This ligament crosses the midsagittal plane of the knee at the level of the tibial insertion of the PCL and attaches to the inferomedial edge of the fabella and the lateral capsule.

**FABELLOFIBULAR LIGAMENT**

The fabellofibular ligament is the most distal edge of the capsular arm of the short head of the biceps femoris. It spans from the lateral edge of the fabella, distally and laterally, to attach to the fibular head just posterior to the attachment of the posterior division of the popliteofibular ligament. If no osseous fabella is present, the ligament’s fibers blend with the anterior fibers of the lateral gastrocnemius tendon, on the posterior aspect of the supracondylar process of the femur, becoming part of the
superficial capsular layer. In a cadaveric study, LaPrade et al found an ossified fabella in 5 of 30 (20%) specimens. In the absence of a bony fabella, some authors have named the structure the short lateral ligament.\(^\text{10,16}\) The fabellofibular ligament is in the greatest tension when the knee is in full extension and is often difficult to identify as it relaxes with knee flexion.\(^\text{5,21}\) Therefore, the clinical significance seems to be important for providing stability of the knee close to full extension. However, biomechanical studies specifically on the fabellofibular ligament have not been performed.

**BIOMECHANICS**

The primary function of the structures of the lateral and posterolateral knee are to resist varus rotation, external tibial rotation, and, to a lesser extent, posterior tibial translation. Several biomechanical studies have shown the FCL, popliteus tendon, and popliteofibular ligament to be the most important stabilizing structures of the posterolateral knee.\(^\text{12,17,22-25}\) The posterolateral structures act in concert with the PCL in providing overall stability of the knee. The complex anatomy of the knee does not allow pure rotational or translational motions to occur. Normal biomechanics of the knee, and abnormal pathomechanics, are the result of complex coupled rotations and translations.

This section discusses posterolateral biomechanics, including anterior-posterior translation, varus-valgus rotation, and internal-external rotation. In addition, the role of intra-articular pressures, osseous configurations, and meniscal configurations will also be addressed.

**OSSEOUS AND MENISCI**

In the majority of the population, the normal mechanical axis of the lower extremity lies slightly medial to the center of the knee. To achieve balance in the stance phase of gait, compressive forces are transmitted through the medial compartment of the knee whereas the lateral structures are under significant tension. During the normal gait cycle, the lateral ligamentous structures of the knee are subjected to greater forces than the medial ligamentous structures and are appropriately more substantial and stronger.\(^\text{11}\)

Likewise, the bony anatomy of the lateral compartment of the knee is quite different from the medial compartment. The medial femoral-tibial compartment has an inherently stable “cup” formation. The convex medial femoral condyle rests in the concavity of the medial tibial plateau. On the contrary, the lateral compartment of the knee is inherently unstable as the convex lateral femoral condyle articulates with a convex lateral tibial plateau. Without additional support these 2 convex surfaces would have minimal surface contact, high contact stresses, and very little stability. The inherently unstable bony geometry of the lateral compartment allows greater motion than the medial compartment, but relies on the posterolateral soft tissue structures to provide the required stability.

The roles of the medial and lateral menisci parallel the roles of the medial and lateral osseous compartments. Both menisci add stability to their respective compartments, but the lateral meniscus must do so when accommodating the relatively greater range of motion of the lateral compartment. The lateral meniscus contributes to lateral knee stability by adding concavity to the lateral tibial plateau. To preserve its increased motion across the tibial plateau, it has less restraining static meniscotibial attachments than the medial meniscus, and is dynamically stabilized by a branch of the popliteus tendon. Because the lateral meniscus is less restrained, it is also less stable than the medial meniscus. This permits it more motion across the tibial plateau with flexion and extension of the knee. The increased excursion of the lateral meniscus limits its ability to compensate for the unstable bony geometry of the lateral tibiofemoral compartment. Therefore, the remaining soft tissues of the PLC must provide significant stability to the lateral side of the knee.\(^\text{25}\)

**ANTERIOR-POSTERIOR TRANSLATION**

The primary restraint to anterior translation of the tibia relative to the femur is the ACL. It accounts for about 86% of the total resistance to anterior tibial translation.\(^\text{11}\) Although the posterior horn of the medial meniscus provides some support to this role, especially in the ACL-deficient knee, it is easily injured when the ACL is compromised. This indicates the minimal amount of support the ACL receives in this role and the necessity of reconstructing it when it is deficient.

Several studies have demonstrated that the posterolateral structures do not prevent primary anterior tibial translation.\(^\text{26,27}\) However, like the posterior horn of the medial meniscus, the PLC helps prevent anterior tibial translation in the ACL-deficient knee. The role of the PLC seems to be more prominent near extension, whereas the role of the posterior horn of the medial meniscus is more noticeable in flexion. This can be demonstrated on physical examination. Typically, an ACL-deficient knee, with an intact posterior horn of the medial meniscus and a torn PLC, will have a markedly positive Lachman test (greater than +2) and a subtle (grade 1) anterior drawer. Conversely, an ACL-deficient knee, with a torn posterior horn of the medial meniscus and an intact PLC, will have a positive anterior drawer and positive Lachman test. However, in the later example, the Lachman test may not be as prominent. Clinically, if an ACL is reconstructed in a knee with a PLC injury, the reconstructed graft will be at higher risk of failure unless the PLC injury is addressed. This underscores the need to thoroughly exam the PLC in all ACL-deficient knees, before reconstruction.

Resistance to posterior tibial translation is far more complex. The stout and centrally located PCL is the primary static stabilizer of the knee and plays a strong role in resisting posterior tibial translation. It provides 95% of the total restraint to posterior tibial displacement forces at all flexion angles.\(^\text{11,22}\)
With the knee in near full extension, isolated sectioning of the posterolateral structures results in increased posterior translation of the lateral tibial plateau. As the knee is flexed to 90 degrees, there is minimal posterior translation. In PCL-deficient knees with transected posterolateral structures, posterior tibial translation of the medial and lateral tibial plateaus is appreciated at 30 and 90 degrees of flexion. Thus, the posterolateral structures are important in resisting posterior tibial translation at small flexion angles (eg, 30 degrees). The PCL and PLC structures work in concert, and injury to both of these structures results in a significant increase in posterior translation of the knee at all flexion angles.

The popliteus is a dynamic stabilizer that assists the PCL by resisting posterior tibial translation. In the intact knee, loading the popliteus has been shown to reduce the in situ forces in the PCL by 9% and 36%, at 90 and 30 degrees of flexion, respectively.29 In the PCL-deficient knee, loading the popliteus has been shown to reduce posterior tibial translation by up to 36%. This amounts to a reduction of posterior translation by approximately 2 to 3 mm. The results of this study confirm that the popliteus muscle complements the PCL in resisting posterior tibial loads and can contribute to knee stability when the ligament is absent.

In a combined PCL and PLC injury model, Harner et al29 demonstrated that a deficiency of the PLC increased posterior tibial translation, in the PCL reconstructed knee, by 4.6 to 6.0 mm. They found a corresponding increase in the in situ forces in the PCL graft of 22% to 150%, if the posterolateral structures were not repaired or reconstructed. These results demonstrate that an isolated PCL graft reconstruction is rendered ineffective and may be overloaded if the posterolateral structures are deficient.

Although the PCL provides the greatest contribution to resisting posterior tibial translation, both biomechanical and clinical studies have shown that it cannot function appropriately in a knee with a deficient PLC. Conversely, clinical studies have demonstrated that many individuals can function with an isolated rupture of the PCL, although their biomechanics may not be identical to that of a healthy knee. These studies demonstrate the importance of a fully functional PLC in resisting posterior tibial translation, and the necessity of repairing or reconstructing the PLC when it is compromised.

VARUS-VALGUS ROTATION

The FCL is the primary restraint to varus stress at all flexion angles of the knee. LaPrade et al30 found that varus forces on the FCL had loading responses from 0 to 90 degrees of flexion, with the greatest being at 30 degrees (12 N/J). Although other soft tissues provide additional support in this function, the FCL receives the least support at 30 degrees of flexion. This makes the FCL most susceptible to injury at 30 degrees of flexion. It has been shown that approximately 300 N of force is required to cause failure of this ligament.19

The PCL is considered a secondary restraint to varus rotation; however, sectioning of this structure with an intact FCL does not affect varus rotation. Conversely, when the PLC is deficient and the PCL is intact, the PCL will provide some resistance to varus stress, but it is not as effective in this role as an intact PLC. In a combined PCL and PLC injury model, Harner et al29 demonstrated that a deficiency of the posterolateral structures after PCL reconstruction increased varus rotation up to 7 degrees. Grood et al27 and Velti et al30 reported that an increase in varus rotation (as much as 19 degrees) can be appreciated with the combined sectioning of the PCL and posterolateral structures. This varus rotation occurred throughout all angles of knee flexion, with the maximal increase observed at 60 degrees.

The posterolateral structures act as the primary restraint to varus rotation especially at lesser degrees of knee flexion (maximal restraint at 30 degrees). The PCL acts as secondary restraint that is most noticeable when the PLC is compromised. The PCL is more effective at resisting varus stresses at greater degrees of knee flexion (60 degrees). To a lesser degree, dynamic resistance to varus rotation is provided by the iliotibial band, biceps femoris muscle tendon complex, and lateral gastrocnemius.

INTERNAL-EXTERNAL ROTATION

The popliteus complex is usually considered the primary restraint to external rotation of the knee. However, recent studies have shown that this function is shared by the FCL. In selective cutting studies, several authors have demonstrated that sectioning of the FCL, popliteus tendon, and deep ligaments of the lateral knee created increases in external rotation at all angles of knee flexion.22,28

Further studies have identified the specific roles of the FCL and the popliteus complex in resisting external rotation. In a recent cadaveric study, LaPrade et al30 found that the FCL resisted greater external rotation forces in the early range of knee flexion (0 to 30 degrees) when compared to the popliteus complex. The external rotation moment responses for the FCL were fairly constant from 0 to 60 degrees of knee flexion, averaging approximately 10 N/J. The mean load response peaked at 30 degrees of flexion (20 N/J) before falling to 8.1 N/J at 90 degrees of flexion.

The popliteus tendon and popliteofibular ligament became more highly loaded by an external rotation torque with higher amounts of knee flexion. The popliteus tendon and the popliteofibular ligament had similar loading patterns to an external rotation torque, from 0 to 90 degrees of knee flexion. Their mean load response generally increased with increasing knee flexion, peaking to approximately 13 N/J at 60 degrees of knee flexion (FCL 10 N/J) before slightly declining to 10 N/J at 90 degrees.30

Therefore, the FCL is the primary restraint to external rotation from 0 to 30 degrees, whereas the

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popliteus complex (popliteus tendon and popliteofibular ligament) assumes a more important primary role in resisting external rotation at knee flexion angles greater than 60 degrees.

The PCL is a secondary stabilizer in resisting external rotation of the tibia. Isolated sectioning of the PCL does not increase external tibial rotation when the PLC is intact. The role of the PCL as a secondary stabilizer becomes significant when the PLC is compromised.

In a PCL-intact knee, isolated sectioning of the posterolateral structures increased external rotation of the lateral tibial plateau, with maximal external rotation (average 13 degrees) demonstrated at 30 degrees of knee flexion. At 90 degrees of flexion, a smaller amount (average 5.3 degrees) of external rotation was observed. Combined sectioning of the PLC and the PCL results in an increase in external rotation of the lateral tibial plateau at all angles of knee flexion, with a maximal increase noted at 90 degrees. As much as 20 degrees of increased external rotation can be appreciated at this degree of knee flexion. This is the basic science behind the external tibial rotation test (or Dial test) commonly used to distinguish between isolated PLC injuries and combined PCL/PLC injuries.

Thus, the fibular collateral ligament is the primary restraint to external rotation at small degrees of knee flexion (30 degrees) and the popliteus complex is the primary restraint against external rotation at greater degrees of knee flexion (60 degrees). The PCL serves as a secondary restraint when the PLC is injured and is most effective at knee flexion angles of 90 degrees.

These basic science studies have important clinical applications. Because isolated sectioning of the PCL does not increase external tibial rotation, any increased tibial external rotation should make a clinician suspicious of a PLC injury. In a combined PCL and PLC injury model, Harner et al. biomechanically demonstrated that a deficiency of the posterolateral structures after PCL reconstruction increased external rotation up to 14 degrees. If the PLC is not reconstructed when the PCL is reconstructed, this resulting increased external rotation places the patient at increased risk of reinjury. Therefore, it is important to reconstruct the posterolateral complex in addition to the PCL to restore normal external tibial rotation restraints.

The posterolateral structures play a small role in preventing primary internal rotation of the knee. Because there is a large amount of variability in internal rotation changes within ACL-intact and ACL-deficient knees, this particular knee instability has not demonstrated much clinical significance.

ARTICULAR CONTACT PRESSURES

The structures of the PLC are important in maintaining joint stability, which can have significant effects on articular contact pressures. Abnormal joint loading patterns and contact pressures can predispose a joint to degeneration. Sectioning the PCL and posterolateral structures has been shown to increase medial, lateral, and patellofemoral compartment contact pressures. In a cadaveric biomechanical model of the knee, Skyhar et al. demonstrated that sequentially sectioning the PCL and then the posterolateral structures resulted in a significant increase in patellofemoral contact pressure at all angles of flexion. This finding is a result of 2 interrelated factors. First, with the loss of these structures, posterior tibial translation increases, thus, diminishing the moment arm of the patellar tendon. The angle between the quadriceps and patellar tendon decreases and creates an increase in the reaction force of the patellofemoral joint. This phenomenon is known as the “reverse Maquet” effect. Second, the quadriceps must exert a greater force because it is acting through a diminished lever arm. This increases articular contact pressures even more.

The loss of ligamentous support creates instability so that the pressure is less likely to be distributed over the articular surface in a normal pattern. A combined PCL and PLC injury induces external rotation of the tibia on the femur, which creates aberrant medial and lateral contact pressures that are already elevated owing to the increase in quadriceps force. Several authors have postulated that this combined injury shifts the center of rotation further into the medial compartment, increasing shear and/or changing contact areas within these compartments during knee motion, ultimately, resulting in a varus-thrust gait pattern. The abnormal contact pressures seen in this combined injury may lead to premature degeneration of the joint and underscore the importance of the PLC in maintaining a healthy knee.

GRAFT RECONSTRUCTIONS

From a clinical standpoint, the PLC can have a significant effect on the survival of the native ACL and PCL. Studies have shown that sectioning of the FCL and surrounding posterolateral structures resulted in increased stresses on the ACL with internal rotation and on the PCL with external rotation.

Similar to the native ACL or PCL, the reconstructed ACL or PCL is at increased risk of failure in a knee with a deficient PLC. Studies have shown that sectioning the FCL and surrounding posterolateral structures results in increased stresses on reconstructed ACL and PCL grafts. LaPrade et al. have shown that coupled loading of varus and internal moments, at 0 and 30 degrees of flexion, significantly increased ACL graft forces. Furthermore, significantly increased forces were demonstrated within PCL grafts with varus and/or coupled external rotation at 30, 60, and 90 degrees of flexion. These studies affirm the recommendation to reconstruct the PLC in both isolated and combined injuries.

REFERENCES


